14.0 OTHER REPRODUCTIVE AND DEVELOPMENTAL STUDIES

STATEMENT TO THE PUBLIC

The DHS reviewers used two different guidelines to evaluate the evidence:

- *Using the guidelines that the International Agency for Research on Cancer uses to assess cancer risks, they considered the evidence as "inadequate" to implicate EMFs. A recent National Institutes of Environmental Health Sciences workgroup reached the same conclusions.*
- *Using the Guidelines developed especially for the California EMF Program, they concluded that they "strongly believe that EMFs do not increase the risk" of reproductive and developmental abnormalities other than miscarriage.*

For use in policy analyses, the DHS reviewers were required to provide a numerical "degree of certainty on a scale from 0 to 100. They represented their best judgment with a little "x" and the range of their confidence with a shaded bar. These are presented below:

CONDITION	REVIE-WER	IARC CLASS	CERTAINTY PHRASE	DEGREE OF CERTAINTY FOR POLICY ANALYSIS THAT AN AGENT (EMFS) INCREASES DISEASE RISK TO SOME DEGREE
Other Reproductive				0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
	1	3	Strongly believe not	
	2	3	Strongly believe not	
	3	3	Strongly believe not	

14.1 THE PATTERN OF EPIDEMIOLOGICAL EVIDENCE

Figure 14.1.1 VDT Studies and Other Reproductive Adverse Effects (not Congenital Anomalies)

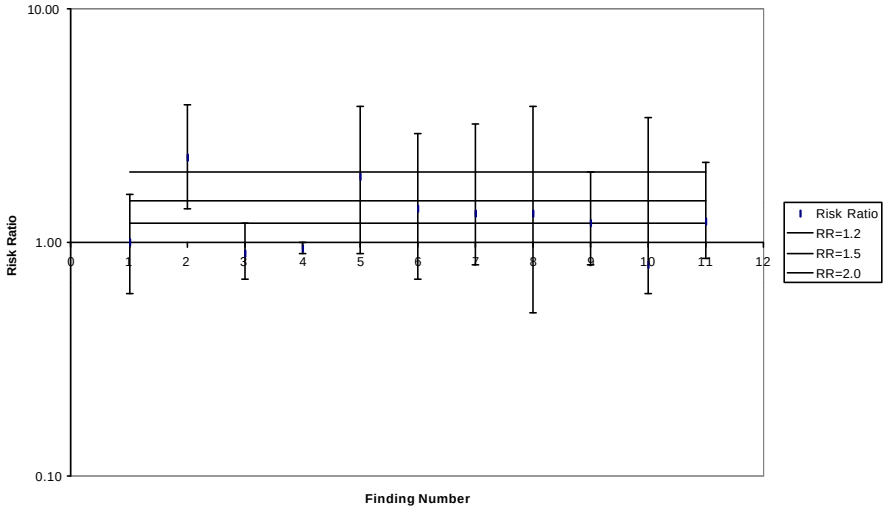

TABLE 14.1.1 STUDIES AND OTHER REPRODUCTIVE ADVERSE EFFECTS (NOT CONGENITAL ANOMALIES)

STUDY NUMBER	REFERENCE	FINDING NUMBER	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
1	(Kurppa, 1985)	1	VDT 4+ hrs/wk	1.00	0.60	1.60
1	(Ericson & Kallen, 1986a)	2	VDT 20+ hrs/wk	2.30	1.40	3.90
3	(Ericson & Kallen, 1986b)	3	VDT High	0.90	0.70	1.20
4	(McDonald et al., 1986)	4	Any VDT use	0.94	0.90	1.00
5	(Westerholm, 1987)	5	VDT, 15 + hrs/wk	1.90	0.90	3.80

TABLE 14.1.1 STUDIES AND OTHER REPRODUCTIVE ADVERSE EFFECTS (NOT CONGENITAL ANOMALIES) [CONT.]

STUDY NUMBER	REFERENCE	FINDING NUMBER	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
6	(Goldhaber et al., 1988)	6	VDT, 20+ hrs/wk	1.40	0.70	2.90
7	(Brandt, 1990)	7	VDT, 31+ hrs /wk	1.32	0.80	3.20
8	(Tikkanen, 1990)	8	VDT, 20+ hrs/wk	1.32	0.50	3.80
9	(Bjerkedal, 1987)	9	Any VDT use	1.20	0.80	2.00
10	(Rodriguez-Pinilla, 1995)	10	Any VDT use	0.80	0.60	3.40
11	(Li, Checkoway & Mueller, 1995)	11	VDT, 45+ hrs/wk	1.23	0.85	2.20

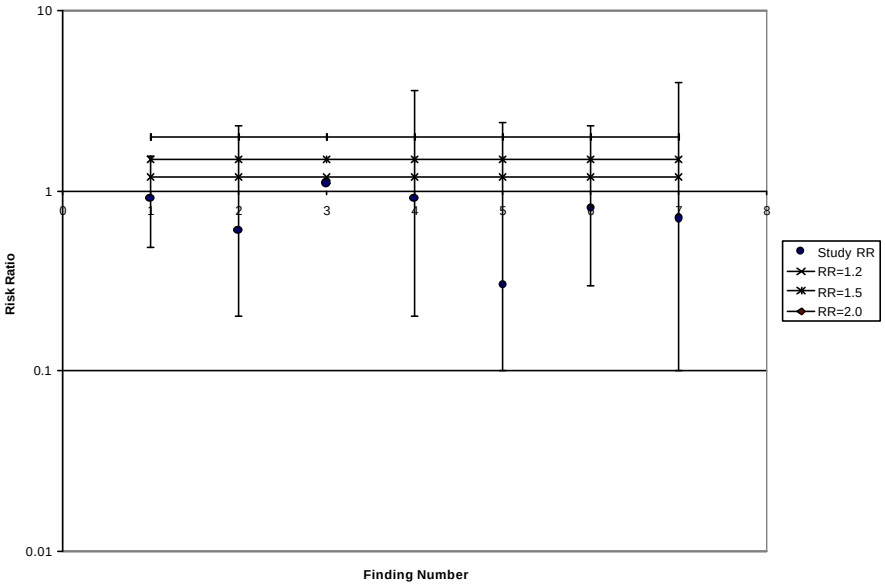

Figure 14.1.2 Residential Studies and Other Reproductive Effects (not Congenital Anomalies)

TABLE 14.1.2 RESIDENTIAL AND OTHER REPRODUCTIVE ADVERSE EFFECTS (NOT CONGENITAL ANOMALIES) STUDIES

STUDY NUMBER	REFERENCE	FINDING NUMBER	OUTCOME	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
1	(Dlugosz et al., 1992)	1	NTD	Electric blanket use	0.9	0.49	1.57
1	(Dlugosz et al., 1992)	2	IUGR	Home spot >1.0 mG cutpoint	0.6	0.2	2.3
1	(Wertheimer & Leeper, 1986)	3	Birthweight<2500	Electric Blanket and Water Bed	1.1	1.1	1.1
2	(Bracken et al., 1995)	4	Birthweight<2500	Home spot >1.0 mG cutpoint	0.9	0.2	3.6
3	(Savitz, 1994)	5	Birthweight<2500	Home spot >0.2 mT cutpoint	0.3	0.1	2.4
3	(Savitz, 1994)	6	Perinatal death	Home spot >0.2 mT cutpoint	0.8	0.3	2.3
3	(Savitz, 1994)	7	Early delivery	Home spot >0.2 mT cutpoint	0.7	0.1	4

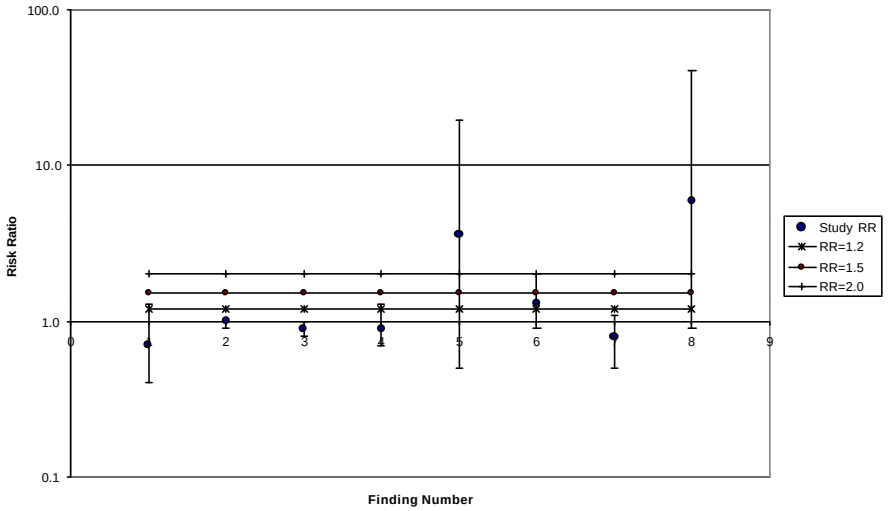

Figure 14.1.3 Occupational Studies and Other Reproductive Effects (not Congenital Anomalies)

TABLE 14.1.3 OCCUPATIONAL AND OTHER REPRODUCTIVE ADVERSE EFFECTS (NOT CONGENITAL ANOMALIES) STUDIES

STUDY	REFERENCE	FINDING NUMBER	OUTCOME	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
1	(Knave et al., 1979)	1	M:F sex ratio	Male EMF occupation	0.70	0.40	1.30
2	(Irgens et al., 1997)	2	M:F sex ratio	Male EMF occupation	1.00	0.90	1.00
2	(Irgens et al., 1997)	3	M:F sex ratio	Female EMF occupation	0.90	0.80	1.00
3	(Tornqvist, 1998)	4	M:F sex ratio	Male EMF occupation	0.90	0.70	1.30
4	(Nordstrom, Birke & Gustavsson, 1983)	5	Perinatal death	Male EMF occupation	3.60	0.50	19.7
3	(Tornqvist, 1998)	6	Perinatal death	Male EMF occupation	1.30	0.90	2.00
3	(Tornqvist, 1998)	7	Birthweight<2500	Male EMF occupation	0.80	0.50	1.10
5	(Buiatti et al., 1984)	8	Male infertility	Male EMF occupation	5.90	0.90	40.2

Figures and Tables 14.1.1-14.1.3 show the reported relative risks of adverse reproductive conditions other than congenital anomalies and spontaneous abortions. Figure 1 and Table 1 are VDT studies. Figure 2 and Table 2 are residential studies. Figure 3 and Table 3 are occupational studies. Overall, there is no pattern of relative risks greater than 1.0, 1.2, or 1.5 for either type of condition or type of exposure. There are about the same number of studies with relative risks above 1.0 and 1.2 as below 1.0 and 1.2 (VDT studies, 7 and 6 out of 11 ($p = 0.16$, $p = 0.23$); residential studies, 7 and 5 out of 12 ($p = 0.19$ for both); occupational studies, 3 out of 8 for both ($p = 0.22$). Very few studies had relative risks above 1.5.

Figure 14.1.4 VDT and Congenital Anomalies Studies

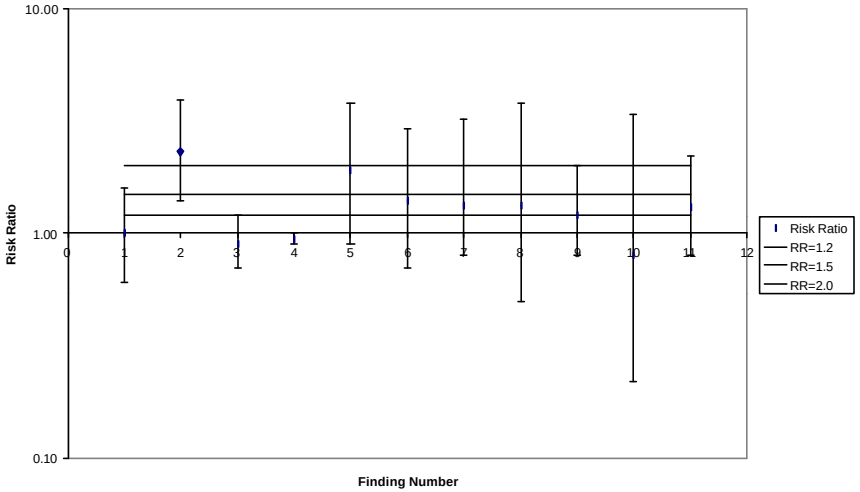

TABLE 14.1.4 VDT AND CONGENITAL ANOMALIES STUDIES

REFERENCE	FINDING NUMBER	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
(Kurppa, 1985)	1	VDT 4+ hrs/wk	1.00	0.60	1.60
(Ericson & Kallen, 1986a)	2	VDT 20+ hrs/wk	2.30	1.40	3.90
(Ericson & Kallen, 1986b)	3	VDT high	0.90	0.70	1.20
(McDonald et al., 1986)	4	Any VDT use	0.94	0.90	1.00
(Westerholm, 1987)	5	VDT, 15+ hrs/wk	1.90	0.90	3.80
(Goldhaber et al., 1988)	6	VDT, 20+ hrs/wk	1.40	0.70	2.90
(Brandt, 1990)	7	VDT, 31+ hrs /wk	1.32	0.80	3.20
(Tikkanen, 1990)	8	VDT, 20+ hrs/wk	1.32	0.50	3.80
(Bjerkedal, 1987)	9	Any VDT use	1.20	0.80	2.00
(Rodriguez-Pinilla, 1995)	10	Any VDT use	0.80	0.22	3.40
(Li et al., 1995)	11	VDT, 45+ hrs/wk	1.30	0.80	2.20

Figure 14.1.5 Residential and Congenital Anomalies Studies

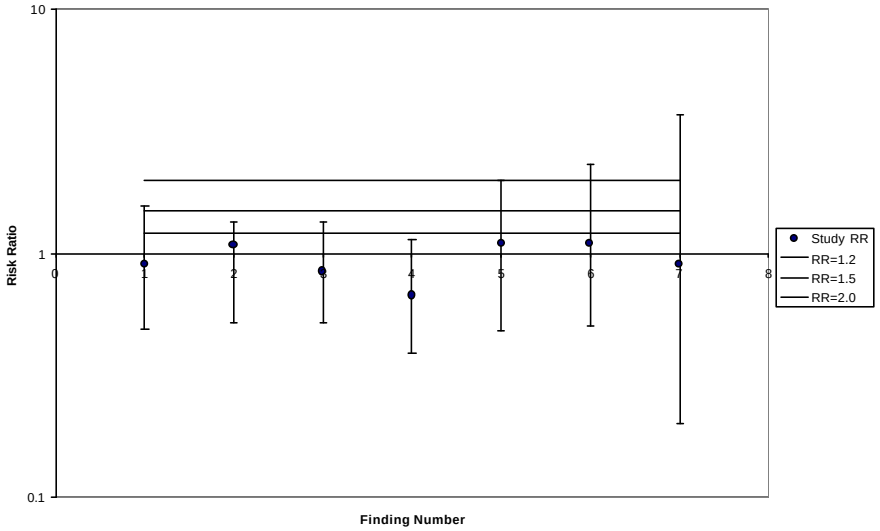

TABLE 14.1.5 RESIDENTIAL CONGENITAL ANOMALIES STUDIES

REFERENCE	FINDING NUMBER	OUTCOME	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
(Dlugosz et al., 1992)	1	NTD	Electric blanket use	0.9	0.49	1.57
(Dlugosz et al., 1992)	2	NTD	Waterbed use	1.08	0.52	1.35
(Dlugosz et al., 1992)	3	Oral cleft	Electric blanket use	0.84	0.52	1.35
(Dlugosz et al., 1992)	4	Oral cleft	Waterbed use	0.67	0.39	1.14
(Milunsky et al., 1992)	5	NTD	Electric blanket use	1.1	0.48	2
(Li et al., 1995)	6	Urinary tract defect	Electric blanket use	1.1	0.5	2.3
(Li et al., 1995)	7	Urinary tract defect	Waterbed use	0.9	0.2	3.7
(Robert et al., 1996)	8	All abnormalities	High voltage lines	0.95	0.45	3.22

Figure 14.1.6 EMF Occupational and Congenital Anomalies Studies

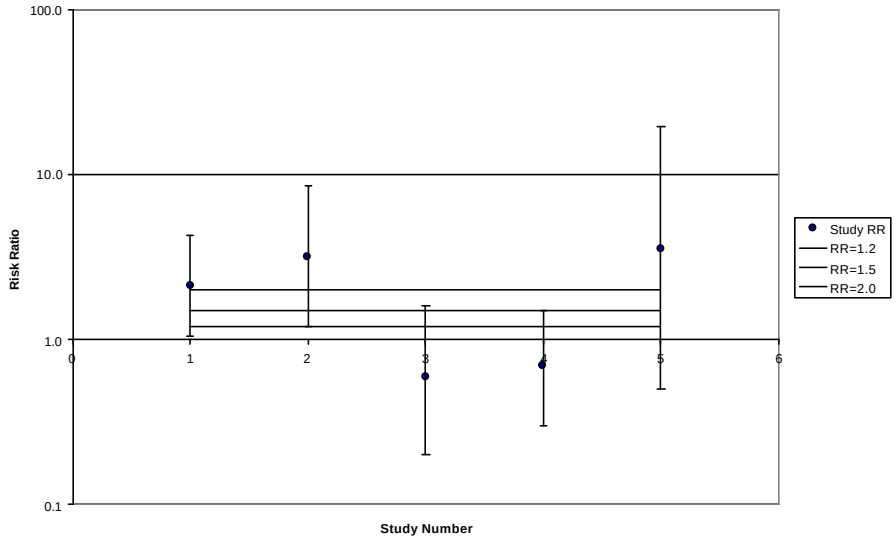

TABLE 14.1.6 OCCUPATIONAL CONGENITAL ANOMALIES STUDIES

STUDY	REFERENCE	FINDING NUMBER	OUTCOME	EXPOSURE	ESTIMATED RISK RATIO	LOWER CL	UPPER CL
1	(Spitz & Johnson, 1985)	1	Congenital Anomalies	Male EMF occupation	2.13	1.05	4.35
2	(Nordstrom et al., 1983)	2	Congenital Anomalies	Male EMF occupation	3.2	1.2	8.6
3	(Bunin et al., 1990)	3	Neuroblastoma	Male EMF occupation	0.60	0.20	1.60
4	(Tornqvist, 1998)	4	Congenital Anomalies	Male EMF occupation	0.70	0.30	1.50
5	(Nordstrom et al., 1983)	5	Perinatal death	Male EMF occupation	3.60	0.50	19.7

Figures and Tables 14.1.4-14.1.6 show the reported relative risks of congenital anomalies. Figure 4 and Table 4 are VDT studies. Figure 5 and Table 5 are residential studies. Figure 6 and Table 6 are occupational studies. Overall, there is no pattern of relative risks greater than 1.0, 1.2, or 1.5 across types of exposure. For the VDT studies, there are about the same number of studies with relative risks above 1.0 and 1.2 as below 1.0 and 1.2 (6 and 5 out of 11; p = 0.23 for both). Only 1 out 11 studies had a relative risk above 1.5. For the residential studies, 3 out of 7 (p = 0.27) had relative risks above 1.0 and no studies had relative risks greater than 1.2. For the occupational studies, the same 3 out of 5 studies had moderate relatives above 1.0, 1.2, 1.5, and 2.0 (p = 0.31).

14.2 ARGUMENTS FOR AND AGAINST CAUSALITY

TABLE 14.2.1 OTHER REPRODUCTIVE AND DEVELOPMENTAL STUDIES

CHANCE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The positive findings are due to chance regardless of the adverse reproductive condition. Only 2 findings out of 31 were significantly above 1.0.	(F1) All four of the electric bed heater findings assessing low birth weight and growth retardation were above 1.0 resulting in a one-sided p-value of 0.06 (Wertheimer & Leeper, 1986), (Bracken et al., 1995).	(C1) Overall, chance cannot be ruled out as an explanation for the observed positive results.

TABLE 14.2.2

BIAS		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Most of the case-control studies are associated with observational bias resulting in the observed positive results.	(F1) Most of the studies used crude assessment of exposure resulting in non-random misclassification and a bias toward the null.	(C1) Non-random misclassification is the major concern resulting in the dilution of an effect, if an effect is present.
(A2) For the positive congenital abnormality studies, only those conditions that were positive may have been presented since a number of conditions were generally assessed.	(F2) There are only two studies that have assessed magnetic fields directly (Savitz, 1994), (Bracken et al., 1995). However, these were not based on personal measures but on area measures resulting in misclassification toward the null.	

TABLE 14.2.3

CONFOUNDING		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Ergonomics and occupational stress from VDT use may have confounded the positive VDT studies.	(F1) It is inappropriate to invoke cofounders that have not been identified; there is no evidence regarding the relationship of VDT use and occupational stress and adverse reproductive conditions.	(C1) Unknown confounders may either bias an association upward or downward. Therefore, no impact.
(A2) If there is an association, it is due to some factor other than EMF related to the surrogate measures used in these studies (such as stress from VDT use or heat from electric bed heater use), since the two studies assessing direct measures (Savitz, 1994), (Bracken et al., 1995) found no associations.	(F2) Confounding was adequately assessed for the few known risk factors of the various endpoints regardless of the main purpose of the study.	(C2) A surrogate measure for EMF such as self-reported electric bed heater use and VDT use may be correlated with another risk factor/exposure unrelated to EMF. However, no such candidates have been adequately identified and explored.
	(F3) Not much can be inferred from the measurement studies since there were only two studies using area measures rather than personal exposures (Savitz, 1994), (Bracken et al., 1995).	

TABLE 14.2.4

STRENGTH OF ASSOCIATION		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) All associations are weak (most are below a relative risk of 1.2) and hence could be due to bias or confounding.	(F1) Non-random exposure misclassification bias is the main problem, which in turns weakens an association if one exists.	(C1) It is possible that non-random misclassification is the reason for the no to very weak associations observed since very crude assessments of exposures were used for all but two studies. The true relative risk may be larger and therefore less vulnerable to bias and confounding.
(A2) The two studies using area magnetic field measures (Savitz, 1994), (Bracken et al., 1995) found a non-significant negative effect to little or no effect where a stronger association is expected.	(F2) Weak, positive associations were found for the overnight magnetic field measurements (Bracken et al., 1995).	(C2) Even evaluating the studies by endpoint, only weak positive associations are observed for those endpoints with more than two studies.
	(F3) Li et al. (Li et al., 1995) found a strong association for urinary tract anomalies and electric blanket users in a subset of women who had a history of sub-fertility	(C3) However, there is a lack of measurement studies to assess if the weak positive studies using surrogate estimates reflect a true association and if the two measurement studies reflect a non-causal relationship. Although very few studies find relative risks above 1.2, this is to be expected.

TABLE 14.2.5

CONSISTENCY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Only the significant associations should be assessed. Overall, out of 52 findings, only 2 studies found significantly positive results for unrelated conditions, a VDT exposure and low birth weight finding (Savitz, 1994) and a paternal occupation and congenital malformation finding (Ericson & Kallen, 1986b).	(F1) There is a slight suggestion of consistency for the electric bed heater studies of low birth weight and growth retardation, as well as VDTs and congenital; but as a group, they are not significantly positive.	(C1) Such inconsistency is expected across very heterogeneous studies.
	(F2) Although the two area measurement studies reported inconsistent results, a consistently positive association may emerge if more area measurement studies were conducted.	(C2) Even for those subgroups where more findings are above 1.0 than below 1.0, chance is a credible explanation of the pattern of evidence.

TABLE 14.2.6

HOMOGENEITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The 2 out of 11 VDT and congenital anomaly studies (Ericson & Kallen, 1986b), (Westerholm, 1987) revealing the largest risks did not restrict analyses to specific phenotypic subgroup, thus increasing the probability these findings are due to chance.	(F1) Due to the considerable heterogeneity of the body of evidence with respect to exposure estimate and endpoint, studies with homogenous endpoints and exposure estimates should be evaluated. For low birth weight and growth retardation, all 4 findings showed relative risks above 1.0 resulting in a low probability ($p = 0.06$) that this is due to chance. Also, for the VDT and congenital anomaly studies, 7 of the 11 findings reported relative risks above 1.0 resulting in a 16% probability of being due to chance.	(C1) Grouping the findings into more homogenous endpoints and/or exposure estimate groups does not reveal any strong consistencies within any of the subgroups.
(A2) In general all the associations are not significant where effects range from weakly protective to weakly negative.	(F2) Some of the VDT and congenital anomalies studies reveal elevated risks. This is to be expected due to the heterogeneous nature of congenital anomalies in terms or their etiology and timing of exposure.	(C2) It is difficult to infer a causal or non-causal association due to the heterogeneity of the group as a whole and the small number of studies available for each individual endpoint.
(A3) The findings with direct exposure measures did not have the strongest relative risks.		

TABLE 14.2.7

DOSE RESPONSE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The VDT studies assessing greater hours of use or "high" use show little or no association.	(F1) The studies using surrogate measures to assess exposure also used very crude assessments of "increased exposure." The assumption of electric bed heaters emitted as a source for high fields and greater hours on a VDT resulting in "more" exposure has not been demonstrated in these and other studies.	(C1) Evidence is lacking to evaluate dose response; most studies did not evaluate risk at various levels of the exposure estimate.

DOSE RESPONSE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A2) Studies assessing electric bed heaters, a source of strong nighttime exposures, found associations close to 1.0.		

TABLE 14.2.8

COHERENCE/VISIBILITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The prevalence of VDT use among working women has increased considerably over time. However, a corresponding increase in adverse reproductive effects is not apparent.	(F1) An apparent increase in adverse reproductive effects with increasing VDT use is not expected due to the heterogeneity of the group, and its association with different etiologies and the lack of sufficient surveillance systems to report these conditions.	(C1) Large, sophisticated studies assessing exposure over time and at the critical time would be needed to address visibility; no such studies have been established.
(A2) A stronger association for studies with direct measures of exposures compared to studies using surrogate measures of exposure was not found.	(F2) There are not enough studies assessing direct EMF measures to evaluate if these exposures result in stronger risks.	
(A3) Among the congenital anomaly studies, one would expect stronger associations for studies focusing on one or two anomalies compared to those studies grouping all anomalies together. The two studies showing the largest elevated risk (Ericson & Kallen, 1986b), (Westerholm, 1987) grouped anomalies.		

TABLE 14.2.9

EXPERIMENTAL EVIDENCE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The results of teratogenic and reproductive effects in mammalian systems are generally negative.	(F1) A number of laboratory studies have reported alterations in the development of chicken embryos exposed to EMF.	(C1) The lack of positive animal studies decrease the confidence only slightly.
	(F2) Animal bioassays of one aspect of a complex mixture are not highly sensitive and may not be linear in risk at high dose resulting in inconsistent and perhaps null results. Null results do not decrease the confidence as much as positive results increase the confidence.	

TABLE 14.2.10

PLAUSIBILITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
No evidentiary base.	No evidentiary base.	(C1) A generally accepted mechanism for biologic effects on reproduction does not currently exits.

TABLE 14.2.11

ANALOGY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See "Generic Issues" chapter.		

TABLE 14.2.12

TEMPORALITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See "Generic Issues" chapter.		

TABLE 14.2.13

SPECIFICITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See "Generic Issues" chapter.		

TABLE 14.2.14

OTHER DISEASE ASSOCIATIONS		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) No biologic reason to consider the associations with other diseases when evaluating the relationship associated with adverse reproductive effects.	(F1) Given that there is an association with spontaneous abortions, it is reasonable to assume that fetuses that are subject to exposure may be damaged even though they survive to term.	(C1) There is some relevance especially with spontaneous abortions.
	(F2) Associations with other diseases will strengthen confidence of causation since EMF is a mixture of components that may influence different biological processes resulting in ill health.	

TABLE 14.2.15

SUMMARY TABLE FOR OTHER REPRODUCTIVE DEVELOPMENTAL CONDITIONS			
	HOW LIKELY IS THIS ATTRIBUTE OF THE EVIDENCE UNDER:		
ATTRIBUTE OF THE EVIDENCE	**"NO-EFFECT" HYPOTHESIS**	**CAUSAL HYPOTHESIS**	**HOW MUCH AND IN WHAT DIRECTION DOES THIS ATTRIBUTE CHANGE CERTAINTY?**
Chance is feasible.	More possible	Possible	Decrease
Bias mainly random misclassification thereby diluting an effect if there is one.	Possible	Possible	No impact
Confounding by unspecified confounders.	Possible	Possible	No impact
Combined chance, bias, and confounding.	More Possible	Possible	Slight decrease
Strength of association (1) not large enough to rule out unspecified bias or confounding.	More possible	Possible	No impact or slight decrease
Consistency: not easily detectable.	More possible	Possible	No impact or slight decrease
Homogeneity: heterogeneous even in similarly grouped endpoints.	More possible	Possible	No impact or slight decrease
Dose response difficult to evaluate due to lacking evidence.	Possible	Possible	No impact
Coherence/visibility difficult to evaluate due to heterogeneous nature of endpoints.	Possible	Possible	No impact
Experimental evidence: animal bioassays are basically negative.	More possible	Possible	No impact or slight decrease
Plausibility: a generally accepted mechanism not defined.	Possible	Possible	No impact
Analogy: see generic discussion.	Possible	Possible	No impact
Specificity: see generic discussion, SAB association.	More possible	Possible	No impact or slight decrease

14.3 POSTERIOR (UPDATED) DEGREE OF CERTAINTY AND IARC CLASSIFICATION

14.3.1 STATEMENTS OF INDIVIDUAL REVIEWERS

Reviewer 1 (DelPizzo)

Degree of Certainty: The human evidence is inconsistent. This reviewer's evaluation of the hypothesis "strongly believe that it is not a cause." For the purpose of decision analysis, Reviewer 1 believes that numerical values of 0 to 10 are appropriate, with the median value to be 5.

IARC Classification: "inadequate" (Class 3).

Reviewer 2 (Neutra)

Degree of certainty: The quality of the exposure assessment in most of the studies of other reproductive outcomes has left a good deal to be desired. The studies have been inconsistent and the pattern is compatible with chance. If the studies had powerful designs, the largely null results would have pulled this reviewer's posterior confidence substantially below the prior, but as it is, the posterior confidence is modestly lower than the prior. Reviewer 2 would characterize the degree of certainty as "Strongly Believe that EMFs do NOT increase the risk of reproductive or developmental problems other than miscarriage to any degree" with a median certainty of 2 and a range from 0.5 to 5.

IARC Classification: The evidence is "inadequate" to implicate EMFs as a reproductive toxicant and would fall in Group 3.

Reviewer 3 (Lee)

Degree of Certainty: The human evidence of the other reproductive and developmental conditions is based on a heterogeneous group of studies with respect to type of condition and exposure assessment making it difficult to evaluate this body of evidence. This reviewer's posterior for a weak relative risk is decreased from her prior by a random association pattern across studies, the heterogeneity of the body of evidence, the fact that bias and confounding cannot be ruled out, and the lack of plausibility evidence. Hence, Reviewer 3's posterior degree of certainty for purposes of the policy analysis falls within the "strongly believe that it is NOT a cause" category with a median value of 5 and a range from 2 to 10.

IARC Classification: The human evidence is inadequate where most studies are susceptible to biases and confounding due to the crude exposure estimates. The overall relative risks are weak where chance cannot be ruled out as an explanation. The heterogeneity of the types of conditions assessed make it difficult to adequately evaluate the causal relationship of any one condition. Hence, exposure is not classifiable and is consistent with Group 3.

14.3.2 SUMMARY OF THE THREE REVIEWERS' CLASSIFICATIONS

CONDITION	REVIE-WER	IARC CLASS	CERTAINTY PHRASE	DEGREE OF CERTAINTY FOR POLICY ANALYSIS THAT AN AGENT (EMFS) INCREASES DISEASE RISK TO SOME DEGREE
Other Reproductive				0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
	1	3	Strongly believe not	
	2	3	Strongly believe not	
	3	3	Strongly believe not	

14.4 QUESTIONS RELEVANT TO DOSE RESPONSE AND POLICY

TABLE 14.4.1

HOW CONFIDENT ARE THE REVIEWERS THAT SPECIFIC EXPOSURE METRIC OR ASPECT OTHER THAN 60 HZ TWA MAGNETIC FIELD IS ASSOCIATED WITH THIS DISEASE?	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
No evidentiary base.	No impact.

TABLE 14.4.2

EVIDENCE FOR THRESHOLD OR PLATEAU	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
No evidentiary base.	No impact.

TABLE 14.4.3

EVIDENCE FOR BIOLOGICAL WINDOWS OF VULNERABILITY	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
No evidentiary base.	No impact.

TABLE 14.4.4

CONSISTENT INDUCTION PERIOD OR REQUIRED DURATION OF EXPOSURE	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
No evidentiary base.	No impact.

TABLE 14.4.5

EMFs COMPARED TO OTHER RISK FACTORS FOR THIS DISEASE	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Hard to evaluate due to the heterogeneity of the group and lack of major risk factors associated with most of the group's endpoints.	None.

TABLE 14.4.6

RELATIVE RISK COMPARED TO THAT WHICH WOULD GENERATE 1/1,000 OR 1/100,000 THEORETICAL LIFETIME RISK	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Lack of evidence to evaluate, but based on the surrogate measure studies, the relative would be very small and not comparable.	No impact.

TABLE 14.4.7

EVIDENCE FOR RACIAL OR CLASS DIFFERENCES IN EXPOSURE OR VULNERABILITY	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
No evidentiary base.	No impact.

TABLE 14.4.8

ROOM FOR IMPROVEMENT IN QUALITY OR SIZE IN BEST EXISTING STUDIES	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) There is considerable room for improvement in the studies published. Future studies should evaluate direct measures of exposure at various levels and timing periods on more homogenous outcome groups, and ascertain potential risk factors as well as other sources of EMF exposures.	(I1) Results from carefully controlled studies assessing at least the more common endpoint would have a considerable impact on policy.

TABLE 14.4.9

NEW STUDIES IN PIPELINE	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
None known to date.	

TABLE 14.4.10

HOW LIKELY IS IT THAT FURTHER STUDIES COULD RESOLVE CONTROVERSIES?	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
See "Room for Improvement" above.	

14.5 CONCLUSIONS OF POLICY-RELEVANT SCIENTIFIC ISSUES

14.5.1 DOSE-RESPONSE ISSUES

The evidentiary base is not sufficient to answer questions about special vulnerabilities, biological windows, thresholds, plateaus, etc.

14.5.2 RESEARCH POLICY

The studies, as a whole, are too heterogeneous with respect to endpoint and exposure assessment to adequately define policy one way or another. It is worth investing in future research for at least the low birth weight and intrauterine growth retardation outcomes due to the positive findings with personal measurements and spontaneous abortions. There is a need for studies—assessing personal exposures from both residential and occupational sources—that are large enough to have the power to evaluate various homogenous subgroups and assess timing of exposure. When exposure conditions are better understood, mechanistic studies should be considered as well since the experimental work to date offers little direction for future epidemiological studies.